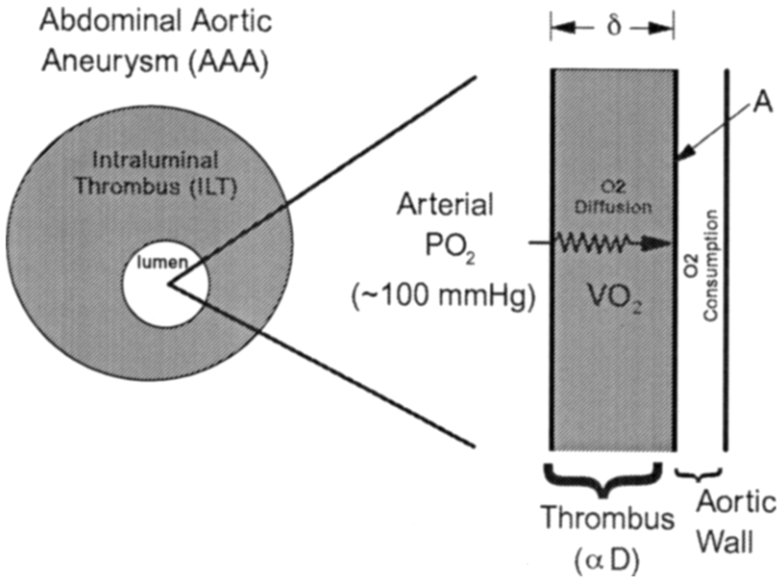

Fig. 1. Oxygen flow rate from lumen to AAA wall.

$\alpha D = (0.024 \text{ ml } O_2/cm^3/760 \text{ mmHg}) \times (2.5 \times 10^{-5} cm^2/s) = 7.9 \times 10^{-10}$ ml O_2/cm/mmHg/s. The drop in oxygen tension across the ILT can be no greater than arterial blood PO_2 (100 mm Hg). Typical ILT thickness in AAA is 1 cm or more. For our example, A = 80 cm^2. With these values, the VO_2 through the ILT from the lumen to the aortic wall is calculated as 6.32×10^{-6} ml O_2/s. Using the gas law conversion of 2.243×10^{-2} ml O_2/μmol, this amounts to 1.69×10^{-2} μmol/min. This represents an O_2 flowrate 20 times less than that required for normal respiration of the SMC in AAA (1.69×10^{-2} μmol/min versus 0.34 μmol/min). The order-of-magnitude analysis presented here indicates that ILT may offer a significant barrier to O_2 transport to the AAA wall. Although our analysis was idealized for simplicity, parameters were chosen to estimate a maximal diffusive flow through the ILT, and none of our choices could likely account for the 20-fold difference between the estimated maximum available oxygen and that needed for normal respiration by the SMC alone in the AAA. We believe that poor diffusion of O_2 through the ILT within AAA causes anoxia, followed by necrosis and diminished resistance by the aortic wall to physiologic distending pressures. This hypothesized effect of ILT may potentially be important in the understanding of the natural history of AAA and we are investigating this further.

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24/41/69501

Regarding "Endovascular arterial intervention: Expression of concern"

To the Editors:

Doctor John M. Porter's observations in his commentary (J VASC SURG 1995;21:995-97) hit the mark. My only disappointment is that he did not specifically address the most pervasive problem: uncontrolled coronary artery interventions performed by invasive cardiologists who self-refer their patients.

As with peripheral endovascular interventions, there is sparce scientific evidence that coronary angioplasty or stent procedures have any long-term benefit, especially as compared with coronary bypass. The same specialists who

properly demanded carefully controlled scientific demonstration of efficacy before reluctantly referring their patients to surgeons seem to place no such demand on themselves with respect to procedures that they perform. That they are able to self-refer their patients for these remunerative procedures makes the practice even more egregious.

On the basis of lessons learned during early attempts at coronary and other small-artery endarterectomies in the 1960s and 1970s, I fear that coronary angioplasty and other coronary intravascular interventions will eventually join Porter's recent list of ineffective procedures that have been used in millions of patients (Table II: Vineberg procedure, chelation therapy, gastric freezing, carotid body excision, extracranial-intracranial bypass, and laser angioplasty). When funding agencies review the costs of coronary intravascular interventions (to say nothing of morbidity), the others will pale by comparison.

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Hypothermia during aneurysm repair: The role of evaporation

To the Editors:

Hypothermia creates morbidity in aneurysm surgery, as Dr. Bush and associates clearly show (Hypothermia during elective abdominal aortic aneurysm repair: the high price of avoidable morbidity. J VASC SURG 1995;21:392-402). Strong support for their study comes from the literature on hypothermia in trauma, liver injuries, and general and pediatric surgery. Jurkovich et al. found that "no (trauma) patient whose core temperature fell below 32° C survived."[1]

Evaporation is often overlooked as a cause of hypothermia. Water evaporates into dry anesthetic gases. Expired air leaves the lungs fully saturated at 37° C. Water also evaporates continually from bowel, yet it remains apparently moist and warm. Water losses carry heat. Every 15 drops of water carries away 0.54 Kcal of heat during evaporation. Preventing evaporation helps prevent hypothermia. A plastic barrier stops loss from the bowel; saturated inspired air at core temperature stops loss from the lung.

In addition to preventing loss, saturated air adds heat to the patient when it is warmed. The lung offers a heat-transfer site superior to skin. A hypothermic lung accepts heat safely from moisture that condenses on its 34° C bronchi from inspired air saturated and warmed to 40° C.

Adding this heat gain to heat conserved by stopping lung evaporation gives a calculated net gain of 10 Kcal per hour (240 Kcal/day!) in a 34° C patient, and gives clinically significant results.[2] Moreover, warmed blood moves directly to the chilled heart, which is wrapped by warm lung. Warming multiplies its effect by speeding metabolism of core organs, adding endogenous heat gain. Other measures, such as warming skin, the room, and all fluids, are effective, yet the hypothermic patient absorbs heat poorly from skin because of vasoconstriction. Warming chilled skin speeds metabolism in poorly oxygenated tissues, which favors creation of acidosis. The benefit of warmed, ***humidified*** air is overlooked if assumptions about heat transfer are based on the insignificant heat capacity of dry air. The latent heat of evaporation and condensation of water is so large that transferring water may increase the heat transfer 100-fold. Water evaporation cools panting dogs, megawatt electrical plants, and, unfortunately, patients under anesthesia.

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